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<u>L2</u>	attention deficit or add or adhd	826990	<u>L2</u>
<u>L1</u>	methylphenidate	1251	<u>L1</u>

END OF SEARCH HISTORY

L6 ANSWER 1 OF 4 DRUGU COPYRIGHT 2003 THOMSON DERWENT  
AN 1985-46909 DRUGU T S  
TI Carbamazepine in Bipolar Illness.  
AU Post R M; Uhde T W  
LO Bethesda, Maryland, United States  
SO Psychopharmacol.Bull. (21, No. 1, 10-17, 1985) 2 Fig. 35 Ref.  
CODEN: PSYBB9 ISSN: 0048-5764  
AV Biological Psychiatry Branch, National Institute of Mental Health, 9000  
Rockville Pike, Bulding 10, R oom 3N-212, Bethesda, MD 20205, U.S.A.  
LA English  
DT Journal  
FA AB; LA; CT  
FS Literature  
AB In a double-blind trial involving over 60 patients with primary affective  
illness, carbamazepine (CBZ) was a useful treatment in some patients who  
were unresponsive to lithium carbonate. 1 Patient responded to CBZ and  
haloperidol, who had not responded to lithium, phenytoin or valproic  
acid. Side-effects which included pruritus, were compared with those of  
lithium. Possible mechanisms of action were considered.  
ABEX. . . rapid onset of a severe manic psychosis on placebo, there was  
improvement in both mania and psychosis and deterioration following  
**dose reduction and placebo**  
**substitution.** 20/37 Patients (54%) with acute depressive  
episodes who entered a double-blind trial, showed mild to moderate  
clinical response. 13/37 (35%).

L6 ANSWER 2 OF 4 DRUGU COPYRIGHT 2003 THOMSON DERWENT  
AN 1984-46899 DRUGU T S  
TI Levodopa Dependence: A Case Report.  
AU Priebe S  
LO Hamburg, Germany, West  
SO Pharmacopsychiatry (17, No. 4, 109-10, 1984) 3 ref.  
CODEN: PHMCDD  
AV Psychiatrische Klinik und Poliklinik der Freien Universitaet Berlin,  
Eschenallee 3, D-1000 Berlin 19, West Germany.  
LA English  
DT Journal  
FA AB; LA; CT  
FS Literature  
AB A female patient who was treated with levodopa p.o. for Parkinson's  
disease developed a dependence on the levodopa as a result of  
experiencing effects of sedation, sleep inducement and analgesia. A  
decarboxylase inhibitor prescribed to help reduce the dose of levodopa  
was ineffective. Previous trials with other parkinsonian agents  
including amantidine were unsuccessful. No extrapyramidal symptoms were  
observed. The patient refused alternative treatment or a reduction in  
levodopa and the course and symptoms of drug withdrawal could not be  
followed as the patient insisted on being discharged.  
ABEX. . . intensity of the parkinsonian symptoms but a relaxing and sedative  
effect was observed. The patient refused alternative treatment or a  
**reduction in dose and placebo**  
**substitution** could not be tried as she discharged herself.

L6 ANSWER 3 OF 4 DRUGB COPYRIGHT 2003 THOMSON DERWENT  
AN 1967-12038 DRUGB T  
TI DIFFERENTIAL EFFECTS OF ABRUPT VERSUS GRADUAL WITHDRAWAL OF  
CHLORPROMAZINE IN HOSPITALIZED CHRONIC SCHIZOPHRENIC PATIENTS.  
AU GREENBERG L M; ROTH S  
LO WASHINGTON, D.C.  
SO AM.J.PSYCHIAT. (123, NO.2, 221-26, 1967)  
DT Journal  
IT CHLORPROMAZINE ABSENCE INFLUENCE ABRUPT-CF. GRADUAL- **DOSAGE-**  
**REDUCTION VIA PLACEBO SUBSTITUTION** IN CHRON.  
SCHIZOPHRENIA BLIND-TEST 42 CASES PSYCHOSEDATIVE PSYCHOSEDATIVES

MENTAL-DISORDER PSYCHOSIS

L6 ANSWER 4 OF 4 ADISNEWS COPYRIGHT (C) 2003 Adis Data Information BV  
AN 1983:602 ADISNEWS ED 8 Aug 2001 UP 8 Aug 2001  
DN 01149954-800501232  
TI ADR news: Withdrawal reactions to long term benzodiazepines.  
SO REACTIONS 29 Jul 1983 ISSN: 0114-9954  
DT (MIX)  
WC 234  
TX. . . after which the drug was gradually withdrawn over 3 months in a  
double-blind manner. Group 1 (18 patients) had their **dose** of  
diazepam **reduced** by half and **placebo**  
**substituted** at the end of 2 weeks, while group 2 (18 patients) had  
a similar dose reduction at 8 weeks. Of. . .

L13 ANSWER 1 OF 3 USPATFULL

AN 87:52069 USPATFULL

TI Counteracting cyclosporin organ toxicity

IN Siegl, Helene, Basel, Switzerland

PA Sandoz Ltd., Basel, Switzerland (non-U.S. corporation)

PI US 4681754 19870721

AI US 1985-771278 19850830 (6)

PRAI GB 1984-22253 19840904

DT Utility

FS Granted

EXNAM Primary Examiner: Friedman, Stanley J.

LREP Sharkin, Gerald D., Honor, Robert S., McGovern, Thomas O.

CLMN Number of Claims: 22

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 566

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Improved cyclosporin therapy in particular for counteracting cyclosporin (e.g. Cyclosporine) organ toxicity, comprises adjunct administration of co-dergocrine.

SUMM . . . . . Klin. Wochenschr. 61, 991-1000 (1983) with Cyclosporine administered i.v. for the first 2-3 days post-transplant and subsequently p.o., plus a **placebo substitute** for co-dergocrine mesylate. Cyclosporine **dosage reduction** is effected in accordance with trough blood levels as measured by RIA. After the first week post transplant the desired. . . .

L13 ANSWER 2 OF 3 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.

AN 83037882 EMBASE

DN 1983037882

TI Treatment of mild and moderate hypertension with medroxalol, an .alpha.- and .beta.-adrenergic antagonist.

AU Schechter P.J.; Tanskanen A.; Tuomilehto J.; Koch-Weser J.

CS Cent. Rech., Merrell Int., 67084 Strasbourg Cedex, France

SO Journal of Cardiovascular Pharmacology, (1982) 4/6 (955-959).

CODEN: JPCDDT

CY United States

DT Journal

FS 037 Drug Literature Index

030 Pharmacology

018 Cardiovascular Diseases and Cardiovascular Surgery

LA English

AB Medroxalol, a new antihypertensive agent with .alpha.- and .beta.-adrenoreceptor blocking properties in both animals and humans, was administered in a single-blind study for 12 weeks to 29 patients with mild and moderate hypertension (standing blood pressure: 188-130/130-100 mm Hg). After 4 weeks of placebo administration, treatment with oral medroxalol was begun. Six weeks later, half the subjects added hydrochlorothiazide, 12.5 mg twice daily, to medroxalol for an additional 6 weeks, and the other half added placebo. During the final 4-week period medroxalol, but not hydrochlorothiazide, was discontinued and **placebo substituted**. Oral medroxalol doses of 100-400 mg twice daily **reduced** standing diastolic pressure to less than 100 mm Hg in 21 of the 26 subjects who completed the study. Compared to the last values on placebo, mean standing blood pressure was decreased by 15.6/12.0 mm Hg during the first 6 weeks of medroxalol at mean daily doses of 388-407 mg. Addition of hydrochlorothiazide permitted some decrease in medroxalol dosage. Upon medroxalol withdrawal, blood pressure and heart rate returned toward pretreatment values, with subjects continuing on diuretic showing lower blood pressures than the untreated individuals. Tolerance to medroxalol, with or without hydrochlorothiazide, was good. Mild orthostatic dizziness was the most frequent complaint associated with therapy, but postural hypotension was not found on

physical examination. Medroxalol appears to be effective and well tolerated for reducing the blood pressure of most patients with mild to moderate hypertension and may be useful for chronic oral therapy of this disease.

AB . . . 6 weeks, and the other half added placebo. During the final 4-week period medroxalol, but not hydrochlorothiazide, was discontinued and **placebo substituted**. Oral medroxalol **doses** of 100-400 mg twice daily **reduced** standing diastolic pressure to less than 100 mm Hg in 21 of the 26 subjects who completed the study. Compared. . .

L13 ANSWER 3 OF 3 ADISNEWS COPYRIGHT (C) 2003 Adis Data Information BV

AN 1999:614 ADISNEWS ED 8 Aug 2001 UP 8 Aug 2001

DN 11738324-800744314

TI Product news: beta-Agonist dose reduction: easy wheezy.

SO INPHARMA 3 Mar 1999 ISSN: 1173-8324

DT (MIX)

WC 328

TX. . . or no dose reduction (18). All patients could use inhaled terbutaline 500microg as required throughout the study. Patients randomised to **dose reduction** had terbutaline **substituted by placebo** in 2 stages, 1 week apart.

**Dose reduction** well-tolerated

After 12 weeks, the median daily terbutaline dose was reduced from 2500microg at week 4 to 500microg in beta-agonist dose. . .

L23 ANSWER 13 OF 48 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.DUPLICATE 5

AN 97172609 EMBASE

DN 1997172609

TI The neurobiology of placebo analgesia: From endogenous opioids to cholecystokinin.

AU Benedetti F.; Amanzio M.

CS F. Benedetti, Dipartimento di Neuroscienze, Universita di Torino, Corso Raffaello 30, 10125 Torino, Italy

SO Progress in Neurobiology, (1997) 52/2 (109-125).

Refs: 165

ISSN: 0301-0082 CODEN: PGNBA5

PUI S 0301-0082(97)00006-3

CY United Kingdom

DT Journal; General Review

FS 002 Physiology

LA English

SL English

AB Placebo is a widespread phenomenon in medicine and biology and its mechanisms are understood only partially. Most of our understanding of placebo comes from studies on pain. In particular, placebo analgesia represents a situation where the administration of a substance known to be non-analgesic produces an analgesic response when the subject is told that it is a painkiller. Several theories try to explain this effect by means of anxiety mechanisms, cognitive processes and classical conditioning. However, the placebo response is bidirectional, i.e analgesic and algesic. In fact, if a subject is told that the ineffective substance is a hyperalgesic drug, a pain increase may occur. The negative effects of placebo are called nocebo and, in extreme cases, they lead to severe pathological conditions. The neurobiology of placebo was born when some authors discovered that placebo analgesia is mediated by endogenous opioids. This claim comes from the observation that the opioid antagonist naloxone can reverse placebo analgesia. On the basis of the discovery of the anti-opioid action of the neuropeptide cholecystokinin, recent studies demonstrate that the blockade of cholecystokinin receptors **potentiates** the **placebo** analgesic response, thus suggesting an inhibitory role of cholecystokinin in placebo analgesia. Thus, by antagonizing the anti-opioid action of cholecystokinin during a **placebo** procedure, a **potentiation** of the endogenous opioid systems can be obtained.

L23 ANSWER 7 OF 48 SCISEARCH COPYRIGHT 2003 THOMSON ISI  
AN 1999:677387 SCISEARCH  
GA The Genuine Article (R) Number: 230ZQ  
TI **Placebos:** Ruse, **potentiator** or powerful therapeutic  
tool?  
AU Gracely R H  
CS NIDCR, CLIN MEASUREMENT & MECHANISMS UNIT, PNMB, NIH, BETHESDA, MD 20892  
CYA USA  
SO JOURNAL OF WOMENS HEALTH & GENDER-BASED MEDICINE, (JUN 1999) Vol. 8, No.  
5, pp. 700-700.  
Publisher: MARY ANN LIEBERT INC PUBL, 2 MADISON AVENUE, LARCHMONT, NY  
10538.  
ISSN: 1524-6094.  
DT Conference; Journal  
FS CLIN; SOCSEARCH  
LA English  
REC Reference Count: 0  
TI **Placebos:** Ruse, **potentiator** or powerful therapeutic  
tool?

L10 ANSWER 4 OF 65 MEDLINE  
 AN 2001531951 MEDLINE  
 DN 21462291 PubMed ID: 11579011  
 TI Pindolol potentiation of paroxetine for generalized social phobia: a double-blind, placebo-controlled, crossover study.  
 AU Stein M B; Sareen J; Hami S; Chao J  
 CS Department of Psychiatry, University of California San Diego, La Jolla, 92093-0985, USA.. mstein@ucsd.edu  
 NC RR-00827 (NCRR)  
 SO AMERICAN JOURNAL OF PSYCHIATRY, (2001 Oct) 158 (10) 1725-7.  
 Journal code: 0370512. ISSN: 0002-953X.  
 CY United States  
 DT (CLINICAL TRIAL)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RANDOMIZED CONTROLLED TRIAL)  
 LA English  
 FS Abridged Index Medicus Journals; Priority Journals  
 EM 200111  
 ED Entered STN: 20011002  
 Last Updated on STN: 20011105  
 Entered Medline: 20011101  
 AB OBJECTIVE: The effectiveness of pindolol as an adjunctive treatment to boost response to selective serotonin reuptake inhibitors (SSRIs) in patients with generalized social phobia was tested. METHOD: A double-blind, placebo-controlled, crossover design was used to compare addition of 5 mg of pindolol t.i.d. or placebo for 4 weeks to a steady paroxetine dose. Subjects were 14 patients with generalized social phobia who were less than "very much improved" on the Clinical Global Impression scale after at least 10 weeks of treatment with a maximally tolerated dose of paroxetine. Changes on the Liebowitz Social Anxiety Scale and the Social Phobia Inventory scores were compared across the two crossover periods. RESULTS: Pindolol was not significantly superior to **placebo for augmenting the effects of** paroxetine on social anxiety symptoms. None of the 14 subjects was deemed a responder to the pindolol arm of the crossover. CONCLUSIONS: Pindolol was no more effective than **placebo in augmenting the effects of SSRI treatment for generalized social phobia.**  
 AB . . . and the Social Phobia Inventory scores were compared across the two crossover periods. RESULTS: Pindolol was not significantly superior to **placebo for augmenting the effects of** paroxetine on social anxiety symptoms. None of the 14 subjects was deemed a responder to the pindolol arm of the crossover. CONCLUSIONS: Pindolol was no more effective than **placebo in augmenting the effects of SSRI treatment for generalized social phobia.**